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In the matter of
Kathleen Matney
Claimant/Survivor
Edeam M. Matney
Deceased

v. Case No. 2001 BLA 0483

Island Creek Coal Co.
Employer

and

Director, Office of Workers' Compensation Programs
Party in Interest

DECISION AND ORDER

AWARDING BENEFITS

This case comes on a request for hearing filed by Mrs. Kathleen Matney[1], widow of Edeam M. Matney[2], pursuant to the provisions of Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended, 30 U.S.C. §§901 *et seq.* (the Act). The Act and implementing regulations, 20 C.F.R. parts 410, 718, and 727 (Regulations), provide compensation and other benefits to:

1. Living coal miners who are totally disabled due to pneumoconiosis and their dependents;
2. Surviving dependents of coal miners whose death was due to pneumoconiosis; and,
3. Surviving dependents of coal miners who were totally disabled due to pneumoconiosis at the time of their death

The Act and Regulations define pneumoconiosis ("black lung disease" or "coal workers pneumoconiosis" or "CWP") as a chronic dust disease of the lungs and its sequelae, including respiratory and pulmonary impairments arising out of coal mine employment.

A hearing was held in Abington, Virginia on July 24, 2001. The Claimant is represented by Joseph Wolfe, Esquire, Grundy, Virginia. Island Creek Coal Corporation (the "Employer") was represented by Mary Rich Maloy, Esquire, Jackson & Kelly, Charleston, West Virginia. An appearance was entered for the Director, OWCP, who did not attend the hearing. Twenty five Director's exhibits (hereinafter "DX") were admitted into evidence without objection. Included within those Director's exhibits are all of the materials

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[1] A/k/a Edith Kathleen Matney, hereinafter the "Claimant" or the "widow".

[2] Hereinafter, the "miner" or the "deceased".

from the Miner's 1985 claim for living miner's benefits[3], including extensive and detailed medical evidence developed between 1985 and 1997. (DX 23). Eight Claimant's exhibits ("CX") were admitted and eleven Employer exhibits (Hereinafter "EX") were admitted. After the hearing the record remained open to receive the parties briefs, which are hereby admitted into evidence.

The Act and Regulations

The Law is set forth by 30 USC §901. *et.seq*. This case involves a claim that is governed by regulations established under the Act at 20 CFR Part 718. Generally, in a Part 718 survivor's claim, a judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. §§ 718.202(a) prior to considering whether the miner's death was due to the disease under §§ 718.205. Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that death will be due to pneumoconiosis if any of the following criteria are met:

> (1) Where competent medical evidence established that the miner's death was due to pneumoconiosis, or
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> (2) Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where the death was caused by complications of pneumoconiosis, or
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> (3) Where the presumption set forth at §§ 718.304 is applicable.
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> (4) However, survivors are not eligible for benefits where the miner's death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneurnoconiosis, unless the evidence establishes that pneumoconiosis was a substantially contributing cause of death.

20 C.F.R. §§ 718.205(c). The term, "substantial contributing cause" under Section 718.205(c)(2) is considered as encompassing situations in which "pneumoconiosis actually hastened the miner's death." ***Shuff v. Cedar Creek Coal Co.***, 967 F.2d 977, 980 (4th Cir. 1992), cert. den. 113 S.Ct. 969 (1993)[4].

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[3]The claim was filed in 1985, denied by a judge in 1988, then heard and denied again in 1991, subjected to modification proceedings and again heard and denied in 1993, again subjected to modification proceedings and denied in 1995, and again subjected to modification proceedings. Judge Daniel Sutton heard the claim and issued a Decision and Order denying benefits in 1997. The Benefits Review Board remanded the claim for reconsideration, and Judge Sutton again concluded that total disability was not established. His February 12, 1999 Decision and Order on Remand Denying Benefits was not appealed and became final.

[4] In denying an award of benefits, "[t]he ALJ concluded that while 'it appears that pneumoconiosis may have hastened ... death,' the fact that Mr. Shuff's death was 'imminent' from the pancreatic cancer, and that 'the cancer itself made Mr. Shuff more susceptible to pneumonia,' meant that pneumoconiosis was not a substantially contributing cause of death." Id. at 979 (emphasis added). The case was remanded for a "definitive finding as to whether the pneumoconiosis actually hastened" the miner's death, citing the conflicting medical evidence and the ALJ's equivocal, inconclusive statement. Id. at 980. In ***Northern Coal Co. v. Director****, OWCP*, 100 F.3d 871 (10th Cir. 1996), the 10rth Circuit adopted the Director's interpretation of the language at 20 C.F.R. §§ 718.205 to mean that a survivor is entitled to benefits if pneumoconiosis hastened the miner's death "to any degree." The United States Court of Appeals for the Third Circuit has held that any condition that hastens the miner's death or has "an actual or

Generally, to receive benefits, a claimant must prove several facts by a preponderance of the evidence. In this case, the parties have stipulated as to the existence of pneumoconiosis. Once the miner is found to have pneumoconiosis, the claimant must show that it arose, at least in part, out of coal mine employment. 20 C.F.R. §§ 718.203(a). If a miner who is suffering from pneumoconiosis was employed for ten years or more in the coal mines, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. 20 C.F.R. §§ 718.203(b). If a miner who is suffering or suffered from pneumoconiosis was employed less than ten years in the nation's coal mines, it shall be determined that such pneumoconiosis arose out of coal mine employment only if competent evidence establishes such a relationship. 20 C.F.R. §§ 718.203(c). Here the miner had 33 years of coal mine employment, and the parties stipulate that the miner's pneumoconiosis arose out of coal mine employment.

"Substantial contributing factor" and "hastens death" are relative measures that may be viewed as part of the Claimant's burden of proof. The assignment of the burden of proof is a rule of substantive law.[5] "Burden of proof," as used in the this setting and under the Administrative Procedure Act[6] is that "[e]xcept as otherwise provided by statute, the proponent of a rule or order has the burden of proof". "Burden of proof" means burden of persuasion, not merely burden of production. 5 U.S.C.A. § 556(d)[7]. The drafters of the APA used the term "burden of proof" to mean the burden of persuasion. ***Director, OWCP, Department of Labor v. Greenwich Collieries*** [Ondecko], 512 U.S. 267, 114 S.Ct. 2251 (1994).[8]

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real share in producing an effect" is a substantially contributing cause of death for purposes of§ 718.205, ***Lukosevicz v. Director, OWCP***, 888 F.2d 1001, 1004 (3d Cir. 1989).

[5] ***American Dredging Co. v. Miller***, 510 U.S. 443, 454, 114 S.Ct. 981, 988, 127 L.Ed.2d 285 (1994)).

[6] 33 U.S.C. § 919(d) ("[N]otwithstanding any other provisions of this chapter, any hearing held under this chapter shall be conducted in accordance with [the APA]"); 5 U.S.C. § 554(c)(2). Longshore and Harbor Workers' Compensation Act ("LHWCA"), 33 U.S.C. §§ 901-950, is incorporated by reference into Part C of the Black Lung Act pursuant to 30 U.S.C. §§ 932(a).

[7] The Tenth and Eleventh Circuits held that the burden of persuasion is greater than the burden of production, ***Alabama By-Products Corp. v. Killingsworth***, 733 F.2d 1511, 6 BLR 2-59 (11th Cir. 1984); ***Kaiser Steel Corp. v. Director, OWCP*** [*Sainz*], 748 F.2d 1426, 7 BLR 2-84 (10th Cir. 1984). These cases arose in the context where an interim presumption is triggered, and the burden of proof shifted from a claimant to an employer/carrier.

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[8] Also known as the risk of nonpersuasion, see 9 J. Wigmore, ***Evidence*** § 2486 (J. Chadbourn rev.1981).

A claimant has the general burden of establishing entitlement *and* the initial burden of going forward with the evidence. The obligation is to persuade the trier of fact of the truth of a proposition, not simply the burden of production, the obligation to come forward with evidence to support a claim.[9] Therefore, the claimant cannot rely on the Director to gather evidence.[10] A claimant, bears the risk of non-persuasion if the evidence is found insufficient to establish a crucial element. ***Oggero v. Director, OWCP***, 7 BLR 1-860 (1985).

Issues

The Employer contested the length of employment, cause of death and the identity of the responsible operator (DX 24). At hearing the issue regarding the idenity of the responsible operator was withdrawn (Tr.,19).

After a review of the evidence of record, I accept that Island Creek Coal Company is the responsible operator.

Evidence

The claim was filed June 27, 2000 (DX 1). Mr. Matney passed away September 3, 1999. He was born August 7, 1920, and was seventy nine years of age when he expired. A death certificate notes that pulmonary embulism/ end stage renal disease were the principal causes of death (DX 6).

Mr. And Mrs. Matney were married April 28, 1942 (DX 7). The Claimant has not remarried.

Medical records from 1999 document lung cancer, heart disease, end stage renal disease, congestive heart failure, and a worsening pulmonary status. (EX1, CX 2,3). The Claimant was a patient at Clinch Valley Medical Center for three days in April, 1999 for pneumonia, chronic obstructive pulmonary disease with acute exacerbation, arteriosclerotic heart disease and chronic renal failure (EX 1, CX 2). From May 4 to June 16, records from Holston Valley Medical Center show treatment for the following:

1. Squamous cell cancer of the lung.
2. Cardiomyopathy.
3. End stage renal disease.
4. Chronic obstructive pulmonary disease/coal workers' pneumoconiosis.
5. Hypertension.
6. Hypercholesterolemia.
7. Weakness; shortness of breath.
8. Congestive heart failure.

Id. Soon after discharge, complaining of increasing worsening of the breathing problems, immediately Mr. Matney returned to the hospital and remained until July 23. The record shows that Dr. Renato Santos

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[9] ***Id***, *also see* ***White v. Director, OWCP***, 6 BLR 1-368 (1983)

[10] ***Id***.

became involved on referral from Dr. H.T. Scott. Id. A report rendered by Dr. Santos on June 6 notes:

1. Worsening pulmonary status. Chronic obstructive pulmonary disease/coal workers' pneumoconiosis.
2. End stage renal disease.
3. Stable cardiac status.
4. Possible left pulmonary nodule.

CX 2. A Dr. Rosser had also been consulted regarding the abnormal chest x-ray. He reviewed the Miner's multiple medical problems. He stated that Mr. Matney had a left lower lobe mass that measured 5 cm in diameter. He thought a CT scan and bronchoscopy would probably be needed. He noted that Mr. Matney reported he had worked about forty (40) years as an underground miner. He said he smoked in the past, but said he had stopped in the 1940*s. Arterial blood gases on oxygen showed PO_2 equal to 101, pCO_2 35.8 and pH 7.49.

Dr. Talton, a radiation oncologist, was consulted. He stated that the cytology from bronchoscopy was positive for poorly differentiated squamous cell carcinoma. He thought it would be possible to irradiate this mass with ports that avoided the heart and planned treatment of 6500 RADS of radiation therapy over sixteen weeks in thirty three doses.

The discharge summary from Holston Valley Medical Center dated June 16 lists squamous cell cancer of the lung, cardiomegaly, end stage renal disease, chronic obstructive pulmonary disease/coal worker*s pneumoconiosis, hypertension, and left ventricular dysfunction with ejection fraction of fifteen per cent (15%). A chest x-ray taken on June 6 displays evidence of cardiomegaly with mild venous hypertension and slight decrease in small effusions. There was a left hilar infiltrate or mass which was unchanged. A chest X-ray of June 24 also showed findings consistent with congestive heart failure and interstitial edema. An echocardiogram performed on June 7 showed mild left ventricular dilation with a severely impaired left ventricle. A resting oxygen saturation on room air during that admission was ninety four percent (94%). A chest x-ray was said to show a 5 cm. left lower lobe mass. A bronchoscopy was performed on June 8. This disclosed a necrotic mass in the left lower lobe. Biopsies were positive for poorly differentiated squamous cancer.

From August 18 to 21, 1999, the Claimant was again at Holston with:

1. Clotted aortic vein graft.
2. Status post right internal jugular perma cath placement by radiology.
3. End stage renal disease, on maintenance hemodialysis.
4. Squamous cell carcinoma of the lung, status post radiation therapy in June 1999.
5. Chronic obstructive pulmonary disease with coal workers' pneumoconiosis.
6. Left ventricular dysfunction with an ejection fraction of fifteen percent..
7. Mitral regurgitation, tricuspid regurgitation.

EX 1. Mr. Matney was discharged to Heritage Hall Nursing Home in Grundy, Virginia, after re-establishing vascular access for dialysis.

On August 31, Mr. Matney was admitted to Buchanan General Hospital with:

1. Confusion.
2. ESRD.
3. Hypertension.
4. History of congestive heart failure.
5. History of lung cancer, non-small-cell in type.

The Miner was readmitted to Buchanan on September 2. He was transferred from the nursing home because of oxygen desaturation beginning in the morning. He had been taking Lovenox and Coumadin. Arterial blood gases on a forty percent venting mask showed a pH of 7.43, PCO_2 was 33 and a $P0_2$ of 72. The patient evidently went rapidly downhill and expired. He was felt to have deep venous thrombosis in the right subclavian vein. Final diagnoses were:

1. Pulmonary embolism.
2. End-stage renal disease.
3. Cerebrovascular accident.
4. Dementia.
5. Hypertension.
6. Arteriosclerotic heart disease.
7. Lung cancer.

EX 1.

Seven hours and twenty minutes after his demise, an autopsy limited to the chest only, was performed at Buchanan by Dr. Joseph Segen, who described the following pertinent gross findings:

General - Body weight: 150 pounds and height: 5'6"

Lungs - Heavy lungs: R- 1350 gm. ; L- 600

- Right lung has four lobes - A 4 cm. tumor with necrotic areas and multiple satellite wound is present in the right upper lobe. The tumor appears to arise from the right upper main bronchus. - "The pleural surfaces and parenchyma contain abundant darkened pigmented nodules." The size of the nodules and their relative percentage of involvement of the pulmonary parenchyma is not mentioned. - Prominently hard hilar lymph nodes, measuring up to 1.5 cm.

Heart - Heart weight: 450 gm. - "The aortic valve leaflets are remarkable for friable vegetations measuring up to 0.5 cm. in greatest dimension." - Thickness of ventricular walls: R- 0.3 cm.; L- 1.3 cm. - Unremarkable myocardium. - Mild coronary arteriosclerosis with less than 20% narrowing of the lumen.

The prosector described the following pertinent microscopic findings:

Lungs - Malignant carcinoma with large and bizarre nuclei, and prominent eosinophilic nucleoli, consistent with Giant-Cell Carcinoma. The tumor shows prominent necrosis, autolysis and micro-calcifications. - "Sub-pleural, septal, interstitial, and peri-vascular fibrosis is present and accompanied by pigment deposition. Dust macules are present and surrounded by focal emphysema in a background of diffuse emphysema. - Moderate number of intra-alveolar pigmented macrophages ("heart failure" cells.) - "Diffuse acute necrotizing

bronchopneumonia." - Hilar lymph nodes are showing "prominent fibrosis" but no tumor.

Heart - Minimal amount of "patchy interstitial fibrosis."

DX 8. Final anatomic diagnoses included:

1. Bilateral bronchopneumonia
2. Giant cell carcinoma with tumor related necrosis
3. Coal workers' pneumoconiosis
4. Cardiomegaly (450 gm.) with changes of heart failure
5. Degeneration of aortic valve with calcification, possible rheumatic in origin
6. Pulmonary edema

Id.

Joshua Perper, M.D., performed an evaluation of the autopsy for the Department of Labor (DX 10). A total of fourteen (14) glass slides taken at autopsy were submitted for examination to Dr. Perper. After a review of the slide material, he determined:

> 1. Mr. Matney had evidence of simple coal workers' pneumoconiosis, with associated centrilobular emphysema and it is likely that the pulmonary cancer could have been also related to exposure to coal mine dust containing silica.
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> 2. Mr. Matney, a former coal miner, developed the coal workers' pneumoconiosis as a result of occupational exposure to mixed coal mine dust containing silica, as demonstrated by the presence in his lungs of anthracotic pigment and silica.
>
> 3. Coal workers' pneumoconiosis with associated centrilobular emphysema was a substantial contributory cause of Mr. Matney's death.

Dr. Perper rendered an opinion that centrilobular emphysema can be caused by exposure to coal mine dust and coal worker*s pneumoconiosis and he concluded that coal worker*s pneumoconiosis was a substantial contributory cause of Mr. Matney*s disability both directly and through the associated centrilobular emphysema through hypoxemia. He also stated that there was a growing body of literature that had substantiated a causal connection between exposure to mixed coal mine dust and coal worker*s pneumoconiosis and the development of lung cancer. Id.

H.T. Scott, who identifies himself as the Miner's treating physician, submitted a report dated June 4, 2001 (CX 1). In the report, Dr. Scott refers to reports issued by Drs. Randy Forehand and Emory Robinette. He notes:

> In my observation over a number of years in treating Mr. Matney, it was my impression that his chronic obstructive pulmonary disease/coal workers' pneumoconiosis was a major factor in his demise and it is my feeling over the last several years the patient was disabled secondary to this pulmonary disease in that he had a history of shortness of breath for a number of years.

Dr. Scott reported symptoms of wheezing; some productive cough in the mornings and required medication for wheezing during this period of time. "When seen in the office he had shortness of breath with very minimal exertion." Id.

In his report dated May 28, 1997, Emory H. Robinette, M.D. recounted that he had originally examined

the Claimant in 1986 (CX 3). On the basis of his original examination, a positive chest x-ray and pulmonary function testing, it was his opinion that the claimant had evidence of underlying Black Lung disease and an impairment of his diffusion capacity of sixty two percent of predicted with mild resting hypoxemia, and that he was disabled from his past coal mine employment (Id., at 3 - 4). He also reported that pulmonary function studies conducted at the time of his 1997 reevaluation revealed an FEV_1 of 1.73 or 91% of predicted, a FVC of 2.85 or 116% of predicted, diffusion capacity slightly impaired at 77% of predicted and elevated lung volume capacity with residual volume. He said that arterial blood gas studies recorded a normal pH (7.465), normal pCO_2 (33.4) and a decreased pO_2 (69.0), consistent with mild resting hypoxemia with a normal spirometry and evidence of air trapping based on lung volume studies (Id., at 3). His impression was:

(1) pneumoconiosis,
(2) resting hypoxemia,
(3) chronic renal insufficiency with associated anemia and
(4) hypertensive cardiovascular disease (Id., at 3).

Dr. Robinette concluded that the Claimant's medical condition had deteriorated over the past several years with increasing subjective dyspnea, and he concluded, "[b]ased on my re-evaluation, I feel that Mr. Matney is disabled from working as an underground coal miner, based on his pulmonary disease alone . . . [o]bviously, he is totally disabled from working on the basis of his multiple medical problems" (Id., at 4).

The Claimant was examined by Dr. Forehand on November 2, 1995 (CX 4). At that time Mr. Matney was seventy five (75) years of age. He reported shortness of breath and wheezing, without cough. He denied ever having smoked cigarettes. An examination of the chest was essentially normal. Pulmonary function studies showed a FVC of 3.50 liters (109%) and a FEV_1 of 2.27 liters (106%). Total lung capacity was 139% and residual volume 145%. Diffusion capacity was eighty eight percent of normal. PO_2 at rest was 65, increasing to 80 after exercise. A chest X-ray was read as 1/0 by Dr. Forehand. After his examination, Dr. Forehand concluded that the claimant had coal worker's pneumoconiosis. He noted that based on normal pulmonary function tests and arterial blood gases with exercise there was no evidence of a totally and permanently disabling respiratory impairment of either a mechanical or gas exchange nature. Id.

The Employer engaged six expert witnesses to rebut Dr. Perper's opinion. Stephen Bush, M.D. reviewed the medical records as well as the autopsy materials from Mr. Matney. After doing so he concluded that the lungs showed evidence of a mild degree of simple coal worker*s pneumoconiosis. He estimated that the coal worker lesions affected an estimated three percent (3%) of the lung substance. He stated that the best preserved slides of lung tissue showed a mild degree of centrilobular emphysema. He felt the findings were most consistent with changes found in individuals with a heavy smoking history such as Mr. Matney had. Dr. Bush opined that the coal worker*s pneumoconiosis was too limited in severity and extent to have contributed to his death. He felt this was supported by the lifetime pulmonary evaluations indicating no significant pulmonary impairment. He determined that Mr. Matney did not suffer from a respiratory impairment as a result of chronic lung disease prior to his death. He also felt that Mr. Matney was totally disabled prior to death as the result of carcinoma of the lung and hypertensive cardiovascular disease and renal impairment. He felt the coal workers' pneumoconiosis or occupational exposure to coal dust did not contribute to respiratory impairment or disability in any way nor did it play a role in or hasten his death.

Dr. Bush reviewed Dr. Perper*s report and pointed out that Dr. Perper appeared to extrapolate to coal miners studies done on silica exposed individuals. He pointed out that although coal mine dust contains some silica, the quantitative differences in exposure are too great to assume that the data for silica can be applied to coal miners. He pointed out that Mr. Matney had a relatively small amount of coal mine dust and silica in his lung sections but was a very heavy cigarette smoker. He disagreed with Dr. Perper regarding any causal relationship between the effects of coal mine dust exposure and death. The available medical evidence indicated that death resulted from carcinoma of the lung and its treatment associated with hypertensive cardiovascular disease. EX 3, EX 6.

Dr. Richard L. Naeye rendered a report on May 5, 2001 He also reviewed the medical records and the autopsy materials. After doing so, he concluded that the characteristic microscopic findings of simple coal worker*s pneumoconiosis were present in the lung tissues of Mr. Matney. He stated that overall the simple coal worker*s pneumoconiosis occupied two little of the whole lung tissue to have caused any disability or hastened death. He asserted that many studies have been undertaken to determine whether occupational exposure to coal mine dust predisposes to lung cancer. No such association has been found when cigarette smoking was taken into consideration and he cited fifteen references to support this statement. He summarized by stating that Mr. Matney had simple coal worker*s pneumoconiosis that was too mild to have contributed in any way to his death. He concluded that coal worker*s pneumoconiosis had no role in causing or hastening the death of Mr. Matney.

Everett Oesterling, M.D., rendered a report dated May30, 2001. He also reviewed the medical records and the autopsy materials. He felt that they showed a mild, micronodular coal worker*s pneumoconiosis with areas of macular change. He felt this level of disease appeared insufficient to have altered pulmonary function to a level that would have resulted in any degree of disability during his lifetime nor would this level of disease in any way have contributed to or hastened his death. He also stated that he would be in sharp disagreement with a comment set forth in the report of Dr. Perper who indicated mine dust is a significant contributing factor to the claimant*s centrilobular emphysema and to his pulmonary malignancy. He stated that workers with advanced silicosis may demonstrate an increased incidence of carcinoma of the lung. However this cannot directly be extrapolated to include a low level of coal worker*s pneumoconiosis as was present in this case. Also this level of coal worker*s pneumoconiosis would not have contributed to his centrilobular pulmonary emphysema. He concluded by stating that coal worker*s pneumoconiosis was not a factor in producing this gentleman*s major lifetime symptomatology nor was it in any way a contributory or hastening factor in his death.

George L. Zalvidar submitted a report dated June 15, 2001 (EX 7). He determined that there is sufficient objective evidence in the record to justify a diagnosis of coal workers' pneumoconiosis. However, he characterized it as a very mild pulmonary impairment " as a result of his smoking habit", which is manifested at autopsy by centrilobular emphysema. He also asserted that smoking also caused the cancer, which he had terminally. According to the report, from a pulmonary standpoint, prior to the discovery of the cancer, at the time of his last examination, was fully capable of performing his usual coal mining work, or work requiring similar exertion. "His pulmonary impairment was too mild to be of any clinical significance." Id.

Kirk Hippensteel, M.D. submitted a report dated June 27, 2001 and was also deposed (EX 8, EX 11). According to Dr. Hippensteel, Mr. Matney would have died at the same time, from the same cause, had

he never been exposed to coal dust or had coal workers* pneumoconiosis, and asserts that his opinion is corroborated by the opinion of several other experts who have reviewed these records. "This man was certainly disabled as a whole man, but from diseases that had no relationship to his prior coal dust exposure, with a reasonable degree of medical certainty." EX 8. Speaking as a pulmonologist, he asserts that a diagnosis of simple coal workers* pneumoconiosis does not necessarily imply that it contributed to death. He asserts that in order to prove so, a functional impairment must be shown:

> Just because centriacinar emphysema is present, does not mean there is functional impairment from it, even if it were stipulated that this centriacinar emphysema was in some way associated with his coal dust exposure rather than his cigarette smoking. Dr. Perper fails to include consideration of the functional data in this man*s case in making his assessment of impairment and hastening of death, while such inclusion is appropriately considered by other pathologists who have reviewed this case.

Id. Dr. Hippensteel examined the Miner in the prior claim on April 24, 1996. He noted that the findings at the time displayed some variability to pulmonary function testing in past, "but showed no permanent ventilatory impairment from any cause including his significant smoking history and significant coal mine dust exposure." Id. He also noted the evidence included some qualifying arterial blood gases at rest "but the extensive records show that this gas exchange impairment was not permanent and also was not associated with impairment during exercise as would occur if his resting gas exchange impairment were related to coal workers* pneumoconiosis....[11] This means that he has no permanent respiratory impairment from any cause that would keep him from working at his last job in the coal mines. The exercise impairment noted in this man can be explained by his heart disease, hypertension, and age, all of which are conditions not caused or contributed to by his coal dust exposure." Id. On October 10, 1996, Dr. Hippensteel was deposed. He testified that the evidence available at that time showed that Mr. Matney had radiographic evidence of coal workers* pneumoconiosis but no specific pulmonary impairment from this disease and that from a respiratory standpoint he could return to his job in the coal mines (DX 23-135).

Thomas Jarboe, M.D. submitted a report dated June 27, 2001 (EX 9). He opined, within reasonable medical probability and/or certainty, that neither coal worker*s pneumoconiosis or the miner*s coal dust exposure played any role in or hastened his death. He determined that pathological findings and the functional findings indicate at most only very mild disease. He disputed Dr. Perper who he says implies that coal dust inhalation may cause lung cancer. "There is no evidence in the medical literature that coal dust inhalation or the presence of pneumoconiosis causes cancer of the lung. I agree with Drs. Bush, Naeye and Osterling that Dr. Perper is in error when he extrapolates the findings in workers exposed to silica to this particular case. There was a minimal amount of silica in Mr. Matney*s pulmonary tissues. It is my reasoned opinion that one cannot assume that his small amount of silica contributed to the development of a lung cancer." Id.

Although Dr. Jarboe agrees with Dr. Perper that coal worker*s pneumoconiosis can be associated with

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[11] He noted that a chest x-ray was consistent with pneumoconiosis (1/0, q/q), that spirometry was normal, that his MVV was mildly reduced with tidal volumes varying by more than 25% indicating inconsistent effort, that lung volumes showed mild air trapping with no restriction and normal diffusion, and that arterial blood gas studies showed minimal hypoxemia with a resting pO2 of 74 (EX 1 at 3). Id.

centrilobular emphysema, he does not accept the final conclusion. He notes that "nearly all workers accept the fact that the amount of emphysema present is proportionate to the amount of dust in the lungs. Dr. Bush has pointed out as have the other pathologists, that the degree of pneumoconiosis in this case was very mild. Dr. Bush stated that it involved only about ten percent of the pulmonary tissues. Since a small amount of dust was present in the lungs, one would not anticipate a significant amount of emphysema as a result." He opines that Mr. Matney died from carcinoma of the lung and ischemic and hypertensive heart disease. Dr. Jarboe notes that the ejection fraction prior to death was only fifteen percent. "This is indicative of very severe cardiac dysfunction. Also, he was undergoing renal dialysis prior to his death. Terminally, he appears to have developed bronchopneumonia which probably was the most immediate cause of death. It is my reasoned opinion that Mr. Matney would have died at the time and of the same causes he did whether or not he had simple coal worker*s pneumoconiosis." Id.

Length of Employment

The claimant bears the burden of establishing the length of his or her coal mine employment. ***Shelesky v. Director, OWCP***, 7 B.L.R. 1-34 (1984); ***Niccoli v. Director, OWCP***, 6 B.L.R. 1-910 (1984); ***Rennie v. U.S. Steel Corp***., 1 B.L.R. 1-859 (1978).

In a hearing before another administrative law judge on March 21, 1991, the Claimant testified that Mr. Matney had worked thirty nine and a half (39.5) years in coal mine employment (DX 23-55). In his application dated November 13, 1985, he alleged thirty nine (39) years (DX 23-1). The Claimant testified that she and the Miner had been married fifty seven (57) years and that the deceased had worked forty (40) years in the mines (Tr., 8-9).

I accept that the Claimant has met her burden on this issue. The Employer has not submitted any evidence to counter this evidence. In fact, the Employer did not dispute this issue in the prior record (DX 23-117 and DX 23-128). A finding concerning the miner's length of coal mine employment may be based exclusively on the claimant's own testimony where it is uncontradicted and credible. ***Bizarri v. Consolidation Coal Co.***, 7 B.L.R. 1-343 (1984); ***Coval v. Pike Coal Co.***, 7 B.L.R. 1-272 (1984); *Gilliam v. G & 0 Coal Co.*, 7 B.L.R. 1-59 (1984).

The Claimant submitted material from the former record (DX 2, DX 23-2). Although not all of the testimony can be substantiated as the alleged work dates are remote (DX 5), a review of the entire record shows that the Miner's testimony is supported by substantial evidence in the record considered as a whole. ***Clayton v. Pyro Mining Co.***, 7 B.L.R. 1-551 (1984); ***Schmidt v. Amax Coal Co.***, 7 B.L.R. 1-489 (1984). After a review of the entire record, I accept that Mr. Matney was credible as to the length of employment.

Therefore, I accept that the Miner had worked thirty nine and a half (39.5) years in coal mine employment.

Cause of Pneumoconiosis

Once the miner is found to have pneumoconiosis, the claimant must show that it arose, at least in part, out of coal mine employment. 20 C.F.R. §§ 718.203(a). If a miner who is suffering from pneumoconiosis was employed for ten years or more in the coal mines, there is a rebuttable presumption that the pneumoconiosis

arose out of such employment. 20 C.F.R. §§ 718.203(b). If a miner who is suffering or suffered from pneumoconiosis was employed less than ten years in the nation's coal mines, it shall be determined that such pneumoconiosis arose out of coal mine employment only if competent evidence establishes such a relationship. 20 C.F.R. §§ 718.203(c). The Claimant has stipulated and the evidence discloses that the Miner had pneumoconiosis and I find that he worked more than thirty nine years in coal mine employment, the Claimant is entitled to the rebuttable presumption on this issue. The burden shifts to the Employer. No evidence to the contrary was submitted. Employer did not contest this issue (See Employer's Pre-hearing Report).

Therefore, I accept that Mr. Matney's pneumoconiosis was caused by coal mine employment.

Evaluation Of the Medical Evidence

It is undisputed that the immediate cause of death in this case includes giant cell carcinoma with tumor, related necrosis, and pulmonary edema. The issue is whether pneumoconiosis was involved. The prosector listed pneumoconiosis as a cause of death in the autopsy report (DX 8). His qualifications are not present in this record. Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. ***Terlip v. Director, OWCP***, 8 B.L.R. 1-363 (1985). Indeed, the Board has held that autopsy reports must be accorded significant probative value regarding the existence and degree of pneumoconiosis because the pathologist who performs the autopsy sees the entire respiratory system as well as other body systems. ***Fetterman v. Director, OWCP***, 7 B.L.R. 1-688, 1-691 (1985).

The death certificate fails to include "pneumoconiosis" as a cause of death; it does refer to pulmonary edema and end stage renal disease (DX 6). It was completed by Aswar Hussain, M.D. A death certificate, in and of itself, is an unreliable report of the miner's condition and it is error for a judge to accept conclusions contained in such a certificate where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner from which to assess the cause of death. ***Smith v. Camco Mining, Inc.***, 13 B.L.R. 1-17 (1989); ***Addison v. Director, OWCP***, 11 B.L.R. 1-68 (1988). The death certificate does not match the autopsy report, which was done only a short time post mortem (DX 8).

None of the evidence shows that cor pumonale is present. I find that the presumption set forth at §§ 718.304 is not applicable. 20 CFR §§718.205(c)(2).

The Employer experts all testified that pneumoconiosis should be ruled out as a cause of death and that other causes led to Mr. Matney's demise. They allege that the record does not show that there was enough pneumoconiosis in the record to have any effect on the Miner. However, simple pneumoconiosis is both legally and medically competent to "hasten" death. See Dr. Naeye's testimony, EX 10, at 22. And Mr. Matney had pneumoconiosis.

Drs. Perper, Dr. Naeye, Bush, and Osterling are all board certified pathologists (DX 10, EX 3-EX 5). Drs. Zalvidar, Jarboe, Hippensteel and Robinette are all board certified in internal medicine and pulmonary

medicine and are NIOSH "B" readers (EX 7-EX 9, CX 8)[12]. Dr. Forehand is board certified in pediatrics and in allergy and immunology and is board eligible in pediatric pulmonary medicine (CX 7). Drs. Santos' and Soctt's qualifications are not part of the record. In making my determination, I must consider the respective qualifications of the physicians. I place no weight on Dr. Forehand's opinions.

I am not bound to accept the opinion or theory of any medical expert, but may weigh the medical evidence and draw its own inferences. ***Kertesz v. Director, OWCP***, 788 F.2d 158 (3d Cir.1986). Moreover, the live miner's claims that Mr. Matney had litigated are not dispositive on the issue whether death was hastened by pneumoconiosis. The prior record shows that the Claimant has proved that he had pneumoconiosis. As of November, 1991, all of the X-ray evidence of record before Administrative Law Judge Robert M. Glennon was positive for pneumoconiosis; but as of that time, the Miner failed to establish total disability (DX 23-61). He submitted a report from Mohammed I. Ranavaya, M.D. and more X-ray readings in a modification attempt to prove that he was totally disabled (DX 23-62). Dr. Ranasvaya deteremined that based on pulmonary function studies and arterial blood gas studies he administered, that the Miner was totally disabled as a result of pneumoconiosis (Id.). Subsequently, the Employer obtained reports from several other physicians and the Claimant was examined by Dr. A. Dahhan. At hearing the Employer conceded the issue regarding pneumoconiosis (See Transcript, DX 23-95), but defended on whether Mr. Matney was totally disabled as a result of pneumoconiosis. After a Decision and Order was entered, by Administrative Law Ludge Robert S. Amery on January 25, 1995, further modification was requested. Dr. Forehand's report (CX 4) was submitted, but in another Decision and Order dated May 25, 1995, the requested modification was denied. After that, the Miner was examined by four physicians. The prior record also contained additional reports from Drs. Abernathy, Sargent and Morgan. Three of these physicians, Drs. Forehand, Hippensteel and Castle, concluded that although simple pneumoconiosis was present, the Claimant did not have any totally disabling respiratory or pulmonary impairment. In addition, seven other non-examining physicians reviewed the body of medical evidence and all concluded that although the Claimant has pneumoconiosis, the record at that time did not demonstrate total disability due to pneumoconiosis (DX 23-150).

Pneumoconiosis substantially contributes to death if it serves to "hasten" death in any way.***Grizzle v. Pickands Mather and Company***, 994 F.2d 1093, 17 BLR 2-123 (4th Cir. 1993); ***Brown v. Rock Creek Mining Co.***, 996 F.2d 812, (6th Cir. 1993); ***Peabody Coal Co. v. Director, OWCP (Railey)***, 972 F.2d 178, 16 BLR 2-121 (7th Cir. 1992); ***Shuff v. Cedar Coal Co.***, 967 F.2d 977, 16 BLR 2-90 (4th Cir. 1992), *cert. denied*, 113 S.Ct. 969 (1993) C.F.R. §§ 718.205(c); ***Lukosevicz v. Director, OWCP***, 888 F.2d 1001, 13 BLR 2-100 (3rd Cir. 1989). In ***Northern Coal Co. v. Director, OWCP***, 100 F.3d 871 (10th Cir. 1996), the court adopted the Director's interpretation of the language at 20 C.F.R. §§ 718.205 to mean that a survivor is entitled to benefits if pneumoconiosis hastened the miner's

[12] A "B-reader" is a physician who has demonstrated proficiency in assessing and classifying x-ray evidence of pneumoconiosis by successful completion of an examination conducted by the United States Public Health Service. 42 C.F.R. 37.51.

death "to any degree." A claimant is eligible for survivor's benefits only after proving that the presence of pneumoconiosis actually shortened the miner's life. 20 C.F.R. §§ 718.205(c)(2); ***Shuff,*** supra; ***Lukosevicz***, supra.

The ***Lukosevicz*** decision examined the Legislative History of that provision in the Act upon which §§ 718.205 is based. It noted that in considering the words governing survivor's claims, Senators Hatch and Congressman Perkins could only agree that the words, "substantially contributing cause," was meant to exclude a Black Lung award to the claimant who was in a traumatic automobile accident causing death, who otherwise merely had pneumoconiosis, but, would allow such benefits to the claimant who had pneumoconiosis, but who died from pneumonia as the stated cause of death. (Id. at 2- 108, 2-109). Therefore, pneumoconiosis does not need to be a proximate cause of death, but must merely contribute to it.[13]

Dr. Perper rendered an opinion that the evidence shows that death was related to coal mine employment:

> It is true that centrilobular emphysema is a known complication of heavy smoking and no information was provided as to whether Mr. Matney was a former smoker. However, as abundantly substantiated in reliable scientific literature in last decades, centrilobular emphysema is a direct result of exposure to mixed coal mine containing silica and coal workers' pneumoconiosis. ....While it is legitimate to recognize the role of smoking in producing centrilobular emphysema, it is equally legitimate to recognize the significant role of exposure to coal mine dust and coal workers' pneumoconiosis, and there is no logical reason to exclude it. As a matter of fact the scientific literature has recognized such significant role of exposure to coal mine dust and centrilobular emphysema, as being significant beyond any effect that may be attributed to smoking.

When asked whether Mr. Matney's coal workers pneumoconiosis a result of his occupational exposure as a coal miner to coal dust, Dr. Perper answered in the affirmative:

> Coal workers' pneumoconiosis is well known to be a result of occupational exposure to coal mine dust. The pathologically findings of Mr. Matney lungs clearly showed that Mr. Matney has pathological evidence at of CWP with clear evidence of silica crystals in pneumoconiotic lesions of the lungs, indicative of exposure to mixed coal dust containing silica.

When asked regarding the cause of death, Dr. Perper listed several:

> ...the autopsy findings clearly revealed that the patients had at least three potential co-existent causes of death namely: the severe acute bronchopneumonia, the lung cancer and the significant

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[13] In ***Kirk v. Director, Office of Workers' Compensation Programs, U.S. Dept. of Labor*** 86 F.3d 1151 [Unpublished Disposition, 1996 WL 273684 (4th Cir. 1996)] the court discussed the hastening standard and whether susceptibility can hasten death. The court determined that it is possible. But given the facts in that case, accepted the administrative law judge's determination that the facts did not warrant it in that case. "Diseases, like jackals on the savanna, kill the weak more readily than the strong".

coal workers' pneumoconiosis with the associated centrilobular emphysema.

Id.

In support of his opinion regarding whether centrilobular emphysema was produced by pneumoconiosis, Dr. Perper cited the following:

> Gregory Wagner, the Director of the Division of Respiratory disease of the National Institute of Occupational Safety and Health clearly states on page 15 of such WHO[14] publication ("Screening and Surveillance of workers exposed to mineral dust" published in 1996 by WHO-Geneva,) "Chronic bronchitis, airflow limitations, CWP[15] and emphysema (italics by G. Wagner) all result from exposure to coal mine dust and may occur in various combination." A 336 pages publication issued in September 1995 by the U.S. Department of Health and Human Services, the Public Health Service, the Centers for Disease Control and Prevention (CDC) and the National Institute for Occupational Safety and Health (NIOSH) entitled "Criteria for a recommended standard - Occupational exposure to coal mine dust" states on page 40 in the chapter on Coal Mine Dust: " COPD[16] refers to three disease processes - chronic bronchitis, emphysema and asthma - that are all characterized by airway dysfunction ..... A major cause of COPD is cigarette smoking; but air pollution and occupational exposure to dust, particularly among smokers, can also cause COPD." On page 51, of the same manual, on page 51, under a section entitled Studies Of COPD in Coal Miners, four American studies published between 1982 and 1992, are quoted as showing that exposure to respirable dust is associated with decrements in lung function among coal miners. Also on page 52, in the section entitled Emphysema, the above publication of the U.S. Department of Health mentions five autopsy studies on U.K. coal miners and two Australian studies between 1982 and 1994 that showed " a significant increase in emphysema among coal miners as compared with non-mining populations." Some of the quoted studies controlled for smoking, and concluded that "the relationship with dust exposures was only apparent among those with centriacinar emphysema" and that "the extent of emphysema in smokers was significantly related to both coal dust of the lungs and to smoking." and "in non smokers, the extent of emphysema was significantly related to both the coal dust content of the lung and age." As a matter of fact the more recent studies concentrate not on the already proven relationship between exposure to coal mine dust and centriacinar (centrilobular) emphysema but on elucidating the mechanism of coal dust toxicity and how it compares to smoking in causing the emphysema. For example, a 1997 report by Canadian researchers, that states "Mineral dust exposure can result in emphysema and chronic airflow obstruction" included both in vitro and in vivo animal experimentation of toxicity of quartz and coal, that support the assumption that dust induced emphysema and smoke induced-emphysema occur through similar mechanisms. Churg A, Katalin, Z and Kevin, L. Mechanisms of Mineral Dust-

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[14] World Health Organization.

[15] Coal workers' pneumoconiosis.

[16] Chronic obstructive pulmonary disease.

induced Emphysema Environ. Health Perspect. 105, Supplement 5, September 1997 (See also Appendix on Coal Workers' Pneumoconiosis and Centrilobular Emphysema).

When asked whether coal workers' pneumoconiosis is a substantial contributory cause of Mr. Matney's disability, and if yes by what mechanism, he replied:

> Coal workers' pneumoconiosis and his morphological substantiation of chronic pulmonary disease was a substantial contributory cause of Mr. Matney's disability both directly and through the associated centrilobular emphysema, through hypoxemia. Furthermore, bronchopneumonia is a known complication of chronic pulmonary disease. Mr. Matney has evidence of insignificant coronary arteriosclerosis and his heart was only slightly to moderately enlarged at 450 gm. Such an enlarged heart could be consistent with primary systemic hypertension. In the absence of clinical data one cannot surmise whether the patient had in addition to that some type of cardiac arrhythmia which might have been aggravated by his chronic pulmonary disease. In addition to that a growing body of literature has substantiated a causal connection between exposure to mixed coal mine dust and coal workers' pneumoconiosis and the development of lung cancer (see appendix II.) Cancer of the lung is known to be related to heavy smoking and is unknown, although possible that Mr. Matney had been a heavy smoker. However, cancer of the lungs has been related in recent years to occupational exposure to silica. The International Association in Cancer Research ([ARC) and OSHA have recognized in recent years that silica is carcinogenic in humans (category 1) and workers' exposed to silica can contract cancer of the lungs. The medical literature of the recent two decades has amply substantiated this position. (See detailed discussion in Appendix.) Coal workers are definitely exposed to coal dust containing silica, and in Mr. Matney case numerous collections of silica crystals were seen in his lung sections. It is true that the association between coal workers' pneumoconiosis is more controversial than that held for silicosis, but a number of recent articles in the scientific literature support the correlation. The clear presence of significant numbers of carcinogenic silica crystals in Mr. Matney' lungs, make such association likely, and does not reasonably justify the exclusion to silica exposure as a pulmonary cancer risk even if Mr. Matney was a former heavy smoker.

Drs. Naeye, Bush and Oesterling found only mild pathological coal workers' pneumoconiosis, and they explained why that neither the pathology, nor the hospitalization records, nor the detailed medical records indicate any association between that pneumoconiosis and the Miner's death. (EX 3, 4, 5, 6) Dr. Naeye confirmed findings which are sufficient for the diagnosis of pneumoconiosis, but he asserted that the abnormalities are nevertheless insufficient to have any clinical significance. (EX 10, at 10-11) .

Although Dr. Naeye testified concerning the thoroughness of the prosector's work, he did not testify that the work was substandard. He stated that they may not be representative of the whole. He said that if the samples were representative, the Miner would have displayed functional problems over the sixteen years his claim had been in process (EX 2). I do not accept this rationale as pneumoconiosis is a progressive disease, and it is competent to manifest itself over time. Even Dr. Naeye concedes that Mr. Matney's slides display pneumoconiosis (Id., DX 10). A more complete analysis is provided later in this decision on this

point. To the contrary, a review of the autopsy report discloses that all quadrants of lung were discussed (DX 8). Dr. Perper related a description of the material in the slides (DX 10). Dr. Osterling agreed that the samples are not representative, however, no evidence was produced to substantiate the allegation . Dr. Osterling referred to several slides that he had reproduced (EX 4). A review of the pictures of slides in the Osterling report to which he directs my attention does not indicate that they are in any way limited to only crucial areas or are substandard in any other way. There is no indication made precisely how the standards were violated, and why the sample is suspect. Neither Dr. Naeye or Dr. Osterling had any objection to the quality of the slides that they had reviewed. An autopsy report should be found in compliance with the quality standards unless there is good cause to believe that the autopsy report is not accurate or that the condition of the miner is being fraudulently represented. ***McLaughlin v. Jones & Laughlin Steel Corp.***, 2 B.L.R. 1-103, 1-108 (1979). I find that the allegation is hollow, and that good cause has not been shown to reject the autopsy. I accept that the autopsy met the standards required by the Act and regulations. 20 CFR §718.106. The Employer did not meet its burden to prove that the autopsy was substandard.

Dr. Perper made the following findings, as pertinent:

> "A": Lung - Pleura shows slight focal fibro-anthracosis. In one place an oval pleural pneumoconiotic micro-nodule with hyaline center is present. The micro-nodule is measuring less than 1 mm. - The pulmonary parenchyma shows two fibro-anthracotic, irregular, mixed type coal dust micronodules, one measuring between 2-3 mm, and the other less than 1 mm. - Slight fairly diffuse anthracotic pigmentation of the inter-alveolar septa, and in peribronchial and perivascular areas. - Slight centrilobular emphysema - Slight to moderate sclerosis of the small intra-pulmonary vessels
>
> "B": Lung - Pleura shows slight focal fibro-anthracosis. No discrete pneumoconiotic lesions present. - Acute bronchopneumonia, severe, extensive - Slight fairly diffuse anthracotic pigmentation of the inter-alveolar septa, and in peribronchial and peri-vascular areas. Small numbers of macrophages containing anthracotic pigment are seen in alveoli. - No micronodules present. In one places a focus of interstitial fibrosis with anthracotic pigmentation, measuring less than 1 mm. - Slight to moderate sclerosis of the small intra-pulmonary vessels. - Slight, focal, centrilobular emphysema with slight focal interstitial fibrosis. - Slight to moderate sclerosis of the small intra-pulmonary vessels
>
> "D": Lung - Pleura with slight focal fibro-arthracosis. In one place a hyaline micronodule measuring less than 1 mm, surrounded by anthracotic pigmentation. - Six (6) fibro-anthracotic micronodules, irregular of the mixed coal dust type, are scattered throughout the lung section, with the larger measuring between 2 and 3 mm, and the others measuring 1 mm or less. Slight scar emphysema is seen around the nodules. - Slight, focal, anthracotic pigmentation of the interalveolar septa, and in peri-bronchial and peri-vascular areas. - Slight centrilobular emphysema. - A tiny focus of acute bronchopneumonia. - Slight to moderate sclerosis of the small intra-pulmonary vessels
>
> "E": Lung - Marked, focal interstitial fibrosis and anthracosis. - Scattered throughout the pulmonary parenchyma are five (5) fibro-anthracotic micronodules of the mixed coal dust type measuring up to 3-4 mm. - Moderate to marked, focal, centrilobular emphysema. - Small foci of acute bronchopneumonia. - Slight to moderate sclerosis of the small intra-pulmonary vessels.

DX 10.

Despite the controversy regarding the validity of the sample, all of the Employer experts conclusively agree that the Miner had "simple" pneumoconiosis. Dr. Naeye testified that there was free silica as well as fibrous tissue in the lungs caused by exposure to coal dust (EX 10, at 17). The black lesions were easy to see (Id., 18). Dr. Naeye testified that simple pneumoconiosis can lead to chronic bronchitis (Id., 22). According to Dr. Naeye, Mr. Matney had episodes of bronchitis over the years, but he didn't develop pneumonia until he developed cancer (Id., 24).

Dr. Naeye discussed all of the pathological abnormalities in the Miner's lungs, including chronic bronchitis (which caused no lung function abnormality), different forms of lung cancer (related to cigarette smoking), "terrible" pneumonia, centrilobular emphysema (which was mild to moderate and caused no significant lung function abnormalities), and coal workers' pneumoconiosis. (Id. at 12-15) Dr. Naeye explained, unequivocally, that the Miner's death was due to kidney failure, and cancer, but not coal workers' pneumoconiosis or industrial bronchitis. (Id. at 16) Similarly, Drs. Bush and Oesterling found only mild pathological coal workers' pneumoconiosis, and they explained why that neither the pathology, nor the hospitalization records, nor the detailed medical records indicate any association between that pneumoconiosis and the Miner's death. (EX 3, 4, 5, 6).

All of the Employer's pathologists found only mild pathological coal workers' pneumoconiosis, and they explained why that neither the pathology, nor the hospitalization records, nor the detailed medical records indicate any association between that pneumoconiosis and the Miner's death. (EX 3, 4, 5, 6). Of course, the prosector, and Dr. Scott, the treating physicians, say otherwise. All of the Employer expert witnesses emphasized the fact that the Miner had been a smoker, smoking approximately forty two (42) pack years, and that cancer was the reason why the Miner acquired pnemonia. They argue that there is not enough coal related material presented to have had any impact on the Miner's health.

The Employer argues that Dr. Perper was incorrect in linking the Miner's centrolobular emphysema without macules to his coal dust exposure (EX 7). It is alleged that Dr. Perper lacked important antemortem information concerning the Miner's physicological data, testing results, smoking histories, and multiple other health problems. (Dr. Zaldivar, EX 7, at 19-20; EX 11 at 15-17) Employer described the "most probative consideration available in this medical record" to refute Dr. Perper:

> It would be pointless to try to go over each and every article that Dr. Perper cited because, ultimately, Mr. Matney was not found to have any respiratory insufficiency by breathing tests prior to his death before the cancer was discovered. Dr. Perper is going far afield from his field in pathology and the description of emphysema on pathological basis to the physiological consequences of emphysema of emphysema regarding airway obstruction. It is well known, and it has been amply demonstrated in the medical literature including textbooks and articles, that the lung has a tremendous reserve. Pathologic lesions of emphysema do not translate well into physiological functioning of the lungs. In this instance whatever centrilobular emphysema may have been found, which in my opinion,

> based on the medical literature, is due to cigarette smoking, did not have any physiological importance. The breathing capacity of Mr. Matney in life was only mildly impaired. This impairment was not translated into any exercise oxygen abnormality. In fact, the oxygen in his blood always improved when he was exercised, which is not a manifestation of physiologically important destruction of lung tissue by emphysema.
>
> Dr. Perper did not have any information regarding the smoking habits of Mr. Matney, which was quite significant spanning a 40-year interval of one pack of cigarettes per day. Nevertheless, he goes on to try to make an argument for his occupation as the cause of cancer.

(EX 7 at 20) I am reminded that Drs. Bush, Naeye and Oesterling also disagreed with Dr. Perper, and explained reasons similar to the reason discussed by Dr. Zaldivar. (EX 5, 6)

I find that the logic is based in large part on the premise that the Miner failed to establish that he was functionally affected by pneumoconiosis by history at the date of death. For example, Dr. Zaldivar advises as fact that the Miner's lungs were "working well." (EX 7). His only basis for this opinion stems frrom testing that was performed two years prior to death. Drs. Jarboe, Hippensteel and Zaldivar, all pulmonologists, all take the position that the record before me does not show that the Claimant had any functional proof of "hastening", in effect setting a requirement that spirometry and blood gas studies are necessary. They assert that unless the pulmonary function studies and arterial blood gas studies show that pneumoconiosis restricts a miner, by reference to the history, the Claimant can not prove "hastening" (EX 7-9; Note Dr. Hippensteel's testimony, EX 11, at 16-20). The Employer pathologists agree. I find this argument irrelevant to Mr. Matney. Pulmonary function studies and arterial blood gas findings made at the time of examination may not accurately reflect the Miner's condition upon his demise. Actually blood gasses performed by Dr. Rosser and at Buchannan Hospital on September 2 are positive, but since they were made during an acute period, they are not very valuable. *See* ***Hess v. Director, OWCP***, 21 B.L.R. 1-141 (1998). There may have been a time lapse when such findings could not have been made. There is proof that the Miner had worsening shortness of breath (CX 1, CX2, EX 1).

Moreover, this argument adds another layer of proof is not required by the law and regulations. 20 CFR §§718.205 (c)sets forth, in part pertinent that proof may include:

> (2) Where pneumoconiosis was a substantially contributing cause or *factor* leading to the miner's death or where the death was caused by *complications of pneumoconiosis*, or

Emphasis added. Case law requires that the "hastening" standard must be applied.

Dr. Robinette performed an examination of the Miner in May, 1997 (CX 3), and Dr. James Castle performed an examination for the Employer in June, 1997 (DX 23-144), more than two years prior to the Miner's death. They performed pulmonary function studies and arterial blood gas studies. They found pneumoconiosis. They also found values on testing beyond the normal reference ranges. Drs. Hippensteel, Jarboe and Zaldivar refute Dr. Robinette's ultimate conclusion. The issue before them at that time was

whether the Miner was totally disabled. On review, Dr. Hippensteel refuted the testing based on diffusion values (EX 11, at 12). In an examination he performed, the gas exchange was also beyond normal. He attributed this to other causes at that time. Note that the testing occurred before the differential diagnosis of cancer was established.

In June, 1999, Dr. Santos listed pneumoconiosis as the chief diagnosis upon referral from Dr. Scott. Dr. Scott noted shortness of breath on minimal exertion (CX 1). He referred Mr. Matney to Dr. Santos because of increasing respiratory problems (CX 2). The observations set forth in Dr. Scott and Dr. Santos' records constitute reports of the Miner's function in mid-1999.[17]

It is not reasonable to extrapolate findings from 1997 to a death that occurred in 1999. It is more reasonable to rely on pathology rather than pulmonology when there are no current findings of record and there has been a reported decline in functioning of record. Moreover to apply the "function" requirement based on history in every case would preclude situations where a combination of "complications" may be involved. See 20 CFR 718.204(c)(2).

Moreover, none of the Employer's experts fully discussed Drs. Santos' and Scott's observations. These observations note an increase in symptoms of shortness of breath. In fact, Dr. Santos' diagnosis was that the Miner had pneumoconiosis causing shortness of breath.(CX 2). In ***Thorn v. Itmann Coal Co.***, 3 F.3d 713 (4th Cir. 1993) discussed "recency". "There may be new or additional evidence developed that discredits an earlier opinion; a comparison of medical reports and tests over a long period of time may conceivably provide a physician with a better perspective than the pioneer physician."The court concluded that "[t]he reasons for crediting such an opinion could be perfectly rational."

All other physicians of record discussed the Miner's significant cardiac disease. Dr. Zalvdivar's report is offered by the Employer to show that the Miner passed away from causes other than pneumoconiosis:

> Terminally, Mr. Matney developed, at an advanced age, cancer of the lung. By that time he was on renal hemodialysis, which is a very debilitating condition in itself. His overall clinical condition was so poor that a physician to whom he was referred for exercise blood gases declined doing them. This was during the last examination by Dr. Robinette.
>
> The autopsy and the clinical records very well document that Mr. Matney had a very large cancer, which was obstructing one lobe of the lung. Someone who had severe heart failure, such as him, with renal failure requiring dialysis and at an advanced age would not be an appropriate surgical candidate. This was recognized by his physicians, who simply recommended radiation therapy. Mr. Matney continues to decline as a result of all these medical conditions, the most serious of which was the cancer and the heart disease. It is

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[17] ***See Richardson v. Perales,*** 402 U.S. 389 (1971).

> important to note that an ejection fraction of 15% is incompatible with any physical activity beyond very limited activities of daily living, but it will prevent the person from doing any walking, or lifting, etc. A 15% left ventricular ejection fraction carries a high mortality; and therefore by itself, this clinical condition of heart was as lethal as the lung cancer. I am enclosing an abstract from an article, which was cited by the electronic textbook, UP-TO-DATE.

(EE 10, at 22-23).

Although pneumoconiosis has been stipulated by the Employer, the progression of Mr. Matney's disease is important in this case. Pneumoconiosis has been established by X-ray as early as October, 1985 (DX 23- 26, DX 23-41). By September 3, 1987, Dr. Zalvidar read that X-ray as positive for pneumoconiosis, with a profusion of 2,2 (DX 23-25). As of the examinations of Dr. Castle in June, 1997 (DX 23-144) and Dr. Emory Robinette in May, 1997, there was no evidence of cancer in the lung (CX 3). Dr. Castle rendered a report that Mr. Matney may have been disabled as a result of hypertensive cardiovascular disease, coronary artery disease, obesity and effects of aging, but these conditions are unrelated to radiographic evidence of coal workers' pneumoconiosis and to his coal mining employment. DX 23-144. Cancer is not noted in the record until after the last physical examination. In fact, the first mention of cancer in the record is from Holston Valley Medical Center in May and June, 1999 (EX 1). The referral to Dr. Santos also occurred at the same time (CX 2). Dr. Santos compared an X-ray he took with one from 1998. A bronchoscopy confirmed cancer was present. There is no record of pulmonary function studies or arterial blood gas studies at that time (Id.). Hypoxemia is present in the record from Buchannan (Id.)

After a review of all of the evidence, I find that the Claimant has met her burden of proof.

I do not attribute significant weight to the fact that there are numerous opinions to the contradict Dr. Perper. The Board has held that a judge is not required to defer to the numerical superiority of x-ray evidence, ***Wilt v. Wolverine Mining Co.***, 14 B.L.R. 1-70 (1990), although it is within his or her discretion to do so, ***Edmiston v. F & R Coal Co.***, 14 B.L.R. 1-65 (1990). I find that, to the contrary, that although each of them provide a conclusion that "pneumoconiosis played no role", the Employer's expert witnesses failed to provide a valid rationale to exclude the possibility that pneumoconiosis is the cause of Mr. Matney's death. A "reasoned" opinion is one in which the judge finds the underlying documentation and data adequate to support the physician's conclusions. ***Fields v. Island Creek Coal Co.***, 10 B.L.R. 1-19 (1987). Indeed, whether a medical report is sufficiently documented and reasoned is for the judge as the finder-of-fact to decide. ***Clark v. Karst-Robbins Coal Co.***, 12 B.L.R. 1-149 (1989)(en banc).

I discount the opinions rendered by Drs. Zalvidar, Hippensteel and Jarboe primarily because they confuse total disability with the "hastening" standard, understate the importance of pathology in establishing causation, and because they require elements of proof based on function testing, such as pulmonary function studies, arterial blood gas studies and diffusion studies. For example, Dr. Jarboe's evaluation is concerned with the "most immediate cause" of death rather than whether pneumoconiosis, which is established in the record is a factor. See Jarboe's report, EX 9. They also did not give significant consideration to the autopsy

and to the time line, especially the recent evidence that the Miner complained of shortness of breath.

Dr. Zaldivar at one time had determined that X-rays showed a diffusion pattern of numerous small opacities (DX 23-25). Later, he failed to consider his former findings, or the effect of time, when he advised as fact that the Miner's lungs were "working well." (EX 7). Moreover, a review of the literature that he submitted shows that it does not impeach those cited by Dr. Perper on the issue regarding the relationship between pneumoconiosis and interlobular emphysema. I note also that hypoxemia was indicated in the Buchannan and Holston records (EX 1, CX 2). According to Dr. Perper, this is consistent with emphysema.

I also accept that Dr. Perper had sufficient information to render his opinion. I discount the opinions of Drs. Naeye, Bush and Osterling in part because they rely on characterizations of the Miner's function as set forth by the pulmonologists' reports. By so doing, they fail to recognize the progressive nature of pneumoconiosis. The opinions are also internally inconsistent. A report may be given little weight where it is internally inconsistent and inadequately reasoned. ***Mabe v. Bishop Coal Co.***, 9 B.L.R. 1-67 (1986). For example, Dr. Naeye also admitted in testimony that simple pneumoconiosis is competent to qualify (EX 10 at 22). If it is enough to qualify, he does not indicate exactly why Mr. Matney's sample exhibits pneumoconiosis, but is not competent to qualify. Also, on one hand, Dr. Naeye included bronchitis as one of the several causes of death (EX 2). In his deposition, he admitted that pneumoconiosis can cause bronchitis (EX 10 at 11). On the other he failed to acknowledge that it is evident in this record. To rationalize these inconsistencies, he notes that pneumoconiosis is "seldom significant" after one quits mining (Id.). Not only is this suspect in this record, it is contrary to the notion that pneumoconiosis is a progressive disease that gets worse over time. ***Eastern Associated Coal Corp. v. Director***, 200 F.3d 250 (4th Cir. 2000). Also see ***Mullins Coal Co. v. Director, OWCP***, *supra*, (describing the etiology of pneumoconiosis as "progressive and irreversible"). In this case bronchitis was significant enough for the Employer's pathologist to list it as a cause of death. As I had alluded earlier, Dr. Naeye testified and by implication, the other Employer pathologists agree, that without continued exposure to coal dust pneumoconiosis seldom progresses. In ***LaBelle Processing Co. v. Swarrow***, 72 F.3d 308 (3d Cir. 1996), the court rejected Employer's reliance on the Surgeon General's Report in support of a finding that coal workers' pneumoconiosis does not progress in the absence of continued exposure. While the Third Circuit noted that the report states that "'[s]imple (coal workers' pneumoconiosis) does not progress in the absence of further exposure,'" it concluded that the report "addressed only the progressive nature of clinical pneumoconiosis." In this vein, the court stated that the legal definition of pneumoconiosis is broader and includes chronic pulmonary diseases such as chronic bronchitis. With regard to chronic bronchitis, the court found "[s]ignificantly, the Surgeon General's Report discusses chronic bronchitis caused by coal dust exposure but at no point suggests that industrial chronic bronchitis cannot progress in the absence of continuing dust exposure." *See also* ***Peabody Coal Co. v. Spese***, 117 F.3d 1001 (7th Cir. 1997) (the Seventh Circuit accepted the Benefits Review Board's rejection of the Surgeon General's report as supportive of the proposition that coal workers' pneumoconiosis does not progress in the absence of

continued exposure). [18] The Board has held that I may discredit the opinion of a physician whose based medical assumptions are contrary to, or in conflict with, the spirit and purposes of the Act. ***Wetherill v. Green Construction Co.***, 5 B.L.R. 1-248, 1-252 (1982).

I also do not accept the Employer's expert testimony because none of the Employer's pathologists offered any facts to lay a foundation for the conclusion that the pneumoconiosis in the record couldn't have hastened death. All of the pathologists used an "either" "or" analogy: none of them considered whether a combination could have occurred. For example, Dr. Osterling asserts that black pigment is suspended within a pink matrix. He notes that these are the start of structures that are a "micronodule of pneumoconiosis". After establishing that it exists pathologically, he states there, in essence, is not enough of it to be a competent cause (EX 4, EX 6). It obviously combined with the pink matter. But the logic does not exclude the net effect. The regulations require consideration whether pneumoconiosis was a substantially contributing cause or *factor*. *Emphasis added*. 20 CFR 718.205(c)(2). More importantly, Dr. Osterling's report identifies the emphysema that Dr. Perper attributes, in part to coal dust exposure.

As with Dr. Naeye, Dr. Bush also refers to the lifetime pulmonology evaluations as proof that pneumoconiosis did not cause death (EX3, EX 6). Dr. Bush opined that the coal worker*s pneumoconiosis was too limited in severity and extent to have contributed to his death. He felt this was supported by the lifetime pulmonary evaluations indicating no significant pulmonary impairment. Again, I do not accept this as more than two years had passed from them to death, and there was evidence that the Miner had increased symptoms after the last spirometry.

I give the autopsy performed by Dr. Segen significant weight. Although his credentials are not of record, I note that autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. ***Terlip v. Director, OWCP***, *supra*. I note that there is substantiation for Dr. Segen's opinion in the medical history of more than thirty nine (39) years of coal mine employment, and the quality and quantity of the yield of material obtained. As stated above, I accept that the autopsy was valid and that the material consisted of macules and emphysema in all four quadrants of the lungs. I also note that Dr. Segen is not employed by the Claimant, the Employer or the Department of Labor. Although I do not consider this to be an important factor, I take it into consideration. ***Melnick v. Consolidation Coal Co.***, 16 B.L.R. 1-31 (1991)(en

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[18] I note that Dr. Naeye gave similar testimony in **Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]**. The court stated that Dr. Naeye based his conclusion on the fact that the miner's "exposure to coal dust had ended in 1973," his pulmonary function study results were normal at that time, and "simple coal workers' pneumoconiosis 'rarely progresses to a more severe disorder if a coal worker quits exposure to mine dust.'" The court disagreed with Dr. Naeye's conclusions to note that he ignored the "assumption of progessivity that underlies much of the statutory scheme" in black lung.

banc).

I accept that Dr. Scott is not as qualified as the Employer expert witnesses, but I accept that he was competent to render his observation regarding increased symptomology. I accept that the Miner's shortness of breath was worse over time. He noted wheezing and a productive cough. As the treating physician, who examined his patient over time, I accept that his opinion is as valid as the three Employer pulmonologists, all of whom relied solely on testing performed more than two years prior to death. The Board has held that, in survivor's claims, "[l]ay testimony constitutes relevant evidence in determining whether claimant has established total respiratory disability pursuant to §§ 718.204(c)." ***Fields v. Island Creek Coal Co.***, 10 B.L.R. 1-19, 1-22 (1987). If lay testimony is relevant, then Dr. Scott's observations are also relevant. His opinion may not carry as much weight as a pathologist, but I attribute some weight to it.

I also credit Dr. Santos' report as to his observations and his diagnosis. The full weight of the evidence substantiates that Mr. Matney had pneumoconiosis as of June, 1999. Although his qualifications are not in evidence, I accept that Dr. Santos was qualified to render treatment at that time, that the treatment was necessary and that treatment was in part to treat shortness of breath.

I credit Dr. Perper's opinions. I must discount the opinions of the Employer pathologists first based on the faulty premise that the Miner must prove by history that he has a functional impairment. For example, Dr. Bush opined that the coal worker*s pneumoconiosis was too limited in severity and extent to have contributed to his death. He felt this was supported by the lifetime pulmonary evaluations indicating no significant pulmonary impairment. I also discount their opinions because they fail to consider the progressive nature of pneumoconiosis. I have discussed Dr. Naeye's opinion that deterioration is "seldom" occurring post employment.

Although the Employer's pathologists attacked the notion that centrilobular emphysema can be caused by pneumoconiosis,[19] it is reasonable that emphysema was a factor that led to death, as was bronchitis. I note that both of these are pulmonary diseases.

I also discount the Employer opinions based on the allegation that the pneumoconiosis exhibited on the slides was insufficient to produce any affect. Even Dr. Naeye admits that bronchitis can be caused by pneumoconiosis. There is no standard or scale that may be used to show how much is "enough". If pneumoconiosis led to the bronchitis diagnosed by the prosector, by Dr. Perper and by Dr. Naeye, it logically "hastened" death. The record reflects that bronchitis occurred intermittently in the living miner's claim.

Although objection was made to Dr. Perper's opinion that emphysema can be caused by pneumoconiosis, I accept Dr. Perper's contention. I note that emphysema may fall under the regulatory definition of

[19] And the pulmonologists asserted that it was not severe enough to have any effect.

pneumoconiosis if they are related to coal dust exposure. ***Robinson v. Director, OWCP***, 3 B.L.R. 1-798.7 (1981); ***Tokarcik v. Consolidation Coal Co.***, 6 B.L.R. 1-666 (1983). In ***Richardson v. Director, OWCP***, 94 F.3d 164 (4th Cir. 1996), the court reiterated that "[c]linical pneumoconiosis is only a small subset of the compensable afflictions that fall within the definition of legal pneumoconiosis under the Act" and that "COPD, if it arises out of coal mine employment, clearly is encompassed within the legal definition of pneumoconiosis, even though it is a disease apart from clinical pneumoconiosis."
The Employer pathologists are correct in noting the Miner also had debilitating kidney disease and heart trouble. I note that these were severe enough to be completely disabling. However, I accept that Dr. Perper is more rational because he presents that pneumoconiosis can adversely affect an already weakened system. He testified that in this case, pneumoconiosis combined with tobacco smoking, to cause development of the lung cancer, which was an established cause of death.

As to qualifications, I accept that Dr. Perper has equivalent credentials to the other pathologists. I do not need to discuss the merits of analytical versus clinical pathology, as I accept that Dr. Perper's logic is more reasonable than those of the other pathologists. I do not accept that the pulmonologists are as qualified as pathologists to evaluate cause of death. As I have commented above, I also discount their opinions based on a failure to accept the progressive nature of pneumoconiosis. I do not give Dr. Perper any special consideration because he was employed by the Department of Labor to evaluate the autopsy report.

Dr. Perper listed three bases for cause of death:

1. The severe acute bronchopneumonia,
2. The lung cancer and ,
3. The significant coal workers' pneumoconiosis with the associated centrilobular emphysema.

If Dr. Perper is correct on any of the three bases, a "hastening" has been proved. Again I note also that hypoxemia was indicated in the Buchannan and Holston records (EX 1, CX 2). This is consistent with Dr. Perper's rendition of the causal events and his theory concerning emphysema. It is logical that Dr. Perper's opinion considers the record evidence that the claimant has shortness of breath that was worsening with time.

Again, a review of the pathology reports shows that pneumoconiosis is present in all of the lobes. I accept that there is dark matter described by Dr. Perper's report[20] and I accept his rendition regarding the quantity and quality of the material. I accept that the record discloses that there was "enough" pneumoconiosis to have an effect cancer and bronchitis.

Dr. Perper sets forth that the pneumoconiosis combined with a history of cigarette smoking to establish the cancer. He set forth articles and medical authority for this proposition. Moreover, a review of the fifteen

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[20] I do not accept Dr. Osterling's demonstrative evidence presentation, and his rendition of the yield.

references cited by Dr. Naeye do not support impeachment of Dr. Perper's opinion. There may be a split of opinion, but I do not accept that Dr. Naeye's view is more rational that Dr. Perper's. Dr. Naeye and Dr. Bush attacked the opinion and the underlying literature, but I accept that Dr. Perper is more logical than either, in part because some of the literature he submitted is the policy of the United States Department of Health and Human Services and the Department of Labor.

For all of the above reasons, Dr. Perper's opinion is entitled to more weight than those of the six Employer expert witnesses. His opinions are more logically reasoned and are substantiated by objective medical evidence. A review of the totality of the evidence supports a finding that the pneumoconiosis caused by Mr. Matney's coal mine employment hastened Mr. Matney's death.

Conclusion

Mrs. Matney has established the presence of pneumoconiosis resulting from her husband's coal mine employment that reasonably hastened his death. Mrs. Matney is therefore entitled to survivor's black lung benefits.

Attorney's Fee

Since I have not received an application from Claimant's attorney for approval of a fee, I do not award attorney's fees at this time. Claimant's attorney has thirty days from receipt of this decision to submit an application for attorney's fees in accordance with 20 C.F.R. §§752.365 and 725.366. With the application, counsel must attach a document showing service of the application upon all parties, including the claimant. The other parties have fifteen days from receipt of the fee application to file an objection to the request. Absent an approved application, no fee may be charged for representation services associated with this claim.

ORDER

It is hereby **ordered** that the claim for benefits filed by Kathleen Matney (a/k/a Edith Kathleen Matney) is hereby **granted**. The Employer, **Island Creek Coal Company** shall:

1. Pay to the Claimant, as survivor of Edeam Matney , all benefits to which she is entitled under the Black Lung Benefits Act, commencing as of September, 1999, the month in which the Miner died;
2. Pay to the Secretary of Labor reimbursement for any payment the Secretary has made to Kathleen Matney under the Act, and to deduct such amounts, as appropriate, from the amount the Employer is ordered to pay under paragraph 1 above;

3. Pay to the Secretary of Labor interest as provided by law under Section 6621 of the Internal Revenue Code of 1954. Interest is to accrue thirty (30) days from the date of the initial determination of entitlement to benefits. 20 C.F.R. §§ 725.608.

4. Claimant's attorney is granted thirty (30) days to submit an application for fees conforming to the requirements of 20 C.F.R. §§ 725.365 and §§ 725.366.

SO ORDERED.

A
Daniel F. Solomon
Administrative Law Judge

Notice of Appeal Rights: Pursuant to 20 C.F.R. §725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date this decision if filed with the District Director, Office of Worker's Compensation Programs, by filing a notice of appeal with the Benefits Review Board, ATTN: Clerk of the Board, Post Office Box 37601, Washington, DC 20013-7601. See 20 C.F.R. §725.478 and §725.479. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits. His address is Frances Perkins Building, Room N-2605, 200 Constitution Avenue, NW, Washington, DC 20210.